AMENDMENT TO THE CLAIMS

This listing of claims will replace all prior versions, and listings, of claims in the applications:

Listing of Claims:

1. (Original) A method of prophylaxis therapy for myocardial infarction (MI) comprising:

selecting a human subject susceptible to MI;

administering to the subject a composition comprising a therapeutically effective amount of an MI therapeutic agent that inhibits leukotriene synthesis *in vivo*, wherein the MI therapeutic agent inhibits leukotriene synthesis by inhibiting the activity of at least one protein selected from 5-Lipoxygenase activating protein (FLAP) and 5-lipoxygenase (5-LO), and

monitoring myeloperoxidase (MPO) level in the human subject before and during the prophylaxis treatment, wherein the MI therapeutic agent is administered in an amount effective to reduce the MPO level in a subject.

2. (Original) A method according to claim 1, further comprising monitoring at least one additional inflammatory marker in the human subject before and during the prophylaxis therapy.

3. (Original) A method according to claim 2, wherein the additional inflammatory marker is C-reactive protein.

4. (Currently amended) A method according to claim 1~~, 2 or 3~~, wherein the monitoring further comprises monitoring a leukotriene level in serum, plasma, or urine from the human subject before and during the prophylaxis treatment, wherein MI therapeutic agent is administered in an amount effective to reduce the leukotriene level in a subject.

5-6. (Canceled)

7. (Currently amended) A method according to ~~any one of claims 1-6~~ claim 1, wherein the MI therapeutic agent inhibits FLAP activity, and the composition is administered in an amount effective to inhibit FLAP polypeptide activity in the human subject.

8-10. (Canceled)

11. (Currently amended) The composition according to claim 7 ~~or 8~~, wherein the MI therapeutic agent comprises BAY-X-1005 or a physiologically acceptable salt, formulation, or pro-drug thereof.

12. (Currently amended) A method according to ~~any one of claims 1-11~~ claim 1, wherein the MI therapeutic agent is administered in an amount effective to reduce the leukotriene level in the subject lower than a median level of leukotrienes in human subjects.

13. (Currently amended) A method according to ~~any one of claims 1-12~~ claim 1, wherein the selecting step comprises selecting a susceptible subject from an elevated measurement of at least one inflammatory marker selected from the group consisting of C-reactive protein (CRP), serum amyloid A, fibrinogen, interleukin-6, tissue necrosis factor-alpha (TNF-alpha), soluble vascular cell adhesion molecules (sVCAM), soluble intervascular adhesion molecules (sICAM), E-selectin, matrix metalloprotease type-1, matrix metalloprotease type-2, matrix metalloprotease type-3, matrix metalloprotease type-9, myeloperoxidase (MPO), and N-tyrosine.

14. (Currently amended) A method according to claim ~~13 or 14~~ 12, wherein the MI therapeutic agent is administered in an amount effective to reduce the elevated serum level of at least one inflammatory marker.

15-17. (Canceled)

18. (currently amended) A method according to ~~any one of claims 1-12~~ claim 1, wherein the selecting comprises selecting a susceptible subject from an elevated measurement of a leukotriene or leukotriene metabolite from the subject.

19-22. (Canceled)

23. (currently amended) A method according to ~~any one of claims 1-12~~ claim 1, where the selecting comprises determining a FLAP genotype or haplotype of a human subject, and selecting for treatment a human subject with a FLAP genotype or haplotype that correlates with an increased risk of myocardial infarction.

24. (Currently amended) A method according to ~~any one of claims 1-12~~ claim 1, wherein the selecting comprises analyzing nucleic acid of a human subject for the presence or absence of at least one 5-lipoxygenase activating protein (FLAP) polymorphism that correlates with a susceptibility to myocardial infarction.

25. (Canceled)

26. (Currently amended) A method according to ~~any one of~~ claims 23, wherein the selecting comprises selecting a human subject having a FLAP (SEQ ID NO: 1) haplotype comprising:

SG13S377 (SEQ ID NO: 1, position 169965), allele A

SG13S114 (SEQ ID NO: 1, position 178096), allele A;

SG13S41 (SEQ ID NO: 1, position 202045), allele A; and

SG13S35 (SEQ ID NO: 1, position 206117), allele G.

27. (Original) A method according to claim 26, wherein the FLAP (SEQ ID NO: 1) polymorphism further comprises SG13S375 (SEQ ID NO: 1, position 164874), allele T.

28. (Currently amended) A method according to claim ~~26 or~~ 27, wherein the presence in said subject of a haplotype further comprised of marker SG13S106 (SNP DG00AAHII) (SEQ ID NO: 1, position 176579), allele G, identifies the subject as having a susceptibility to MI.

29. (Currently amended) A method according to claim ~~26 or~~ 27, wherein the presence in said subject of a haplotype further comprised of marker SG13S106 (SNP DG00AAHII) (SEQ ID NO: 1, position 176579), allele G; SG13S30 (SEQ ID NO: 1, position 193840), allele G; and SG13S42 (SEQ ID NO: 1, position 203877), allele A, identifies the subject as having a susceptibility to MI.

30. (Original) A method of prophylaxis therapy for myocardial infarction (MI) comprising:

selecting a human subject having a FLAP (SEQ ID NO: 1) haplotype comprising: SG13S375 (SEQ ID NO: 1, position 164874), allele T;

administering to the subject a composition comprising a therapeutically effective amount of an MI therapeutic agent that inhibits leukotriene synthesis *in vivo*, wherein the MI therapeutic agent inhibits leukotriene synthesis, and

monitoring myeloperoxidase (MPO) level in the human subject before and during the prophylaxis treatment, wherein the MI therapeutic agent is administered in an amount effective to reduce the MPO level in a subject.

31. (Original) A method according to claim 30, wherein the selecting comprises selecting a human subject having a FLAP (SEQ ID NO: 1) haplotype further comprising:

SG13S25 (SEQ ID NO: 1, position 165553), allele G;

SG13S32 (SEQ ID NO: 1, position 176579), allele A; and

SG13S106 (SEQ ID NO: 1, position 198547), allele G or A.

32. (Currently amended) A method according to any one of claim 30 ~~or 31~~, wherein the presence in said subject of a haplotype further comprised of markers:

SG13S25 (SEQ ID NO: 1, position 165553), allele G;

SG13S99 (DG00AAFIU), allele T (SEQ ID NO: 1, position 138551);

SG13S377 (DG00AAJFF) (SEQ ID NO: 1, position 169965), allele G;

SG13S106 [SNP DG00AAHII] (SEQ ID NO: 1, position 176579), allele G;

SG13S32 (SEQ ID NO: 1, position 198547), allele A; and

SG13S35 (SEQ ID NO: 1, position 206117), allele G,

identifies the subject as having a susceptibility to MI.

33. (Currently amended) A method according to claim [[23]] 30, wherein the presence in said subject of a haplotype comprised of markers:

SG13S377 (SEQ ID NO: 1, position 169965), allele A;

SG13S114 (SEQ ID NO: 1, position 178096), allele A;

SG13S41 (SEQ ID NO: 1, position 202045), allele A; and

SG13S35 (SEQ ID NO: 1, position 206117), allele G,

identifies the subject as having a susceptibility to MI.

34. (Currently amended) A method according to claim [[23]] 30, wherein the presence in said subject of a haplotype comprised of markers:

SG13S375 (SEQ ID NO: 1, position 164874), allele T

SG13S25 (SEQ ID NO: 1, position 165553), allele G;

SG13S32 (SEQ ID NO: 1, position 176579), allele A; and

SG13S106 (SEQ ID NO: 1, position 198547), allele G or A,

identifies the subject as having a susceptibility to MI.

35-36. (Canceled)

37. (Previously presented) A method of prophylaxis therapy for myocardial infarction (MI) comprising:

analyzing nucleic acid of a human subject for the presence and absence of a FLAP haplotype, wherein the haplotype is comprised of markers:

SG13S375 (SEQ ID NO: 1, position 164874), allele T;

SG13S25 (SEQ ID NO: 1, position 165553), allele G;

SG13S32 (SEQ ID NO: 1, position 176579), allele A; and

SG13S106 (SEQ ID NO: 1, position 198547206117), allele G or A,

selecting for treatment a human subject having nucleic acid with the presence of the FLAP haplotype,

administering to the subject a composition comprising a therapeutically effective amount of an MI therapeutic agent that inhibits leukotriene synthesis *in vivo*, wherein the MI therapeutic agent inhibits leukotriene synthesis.

38. (Original) A method according to claim 37, comprising monitoring at least one inflammatory marker in the human subject before and during the prophylaxis therapy.

39-42. (Canceled)

43. (Currently amended) A method according to ~~any one of claims 37-42~~ claim 37, wherein the MI therapeutic agent inhibits FLAP activity or 5-LO activity.

44. (Canceled)

45. (Currently amended) A method according to ~~any one of claims 37-44~~ claim 37, wherein the MI therapeutic agent is administered in an amount effective to reduce the leukotriene level in the subject lower than a median level of leukotrienes in human subjects.

46. (Currently amended) A method of prophylaxis for myocardial infarction (MI) comprising:

administering to a subject in need of prophylaxis for myocardial infarction ~~infraction~~ a composition comprising a therapeutically effective amount of an MI therapeutic agent that inhibits leukotriene synthesis *in vivo*, and

monitoring myeloperoxidase (MPO) level in the human subject before and during the prophylaxis treatment, wherein the MI therapeutic agent is administered in an amount effective to reduce the MPO level in a subject.

47. (Original) A method of screening a human subject for risk of developing myocardial infarction, comprising:

contacting a blood sample from the human subject with a calcium ionophore to stimulate production of a leukotriene; and

measuring production of a leukotriene in the blood sample after the contacting step, wherein elevated leukotriene production compared to a control correlates with increased risk of developing myocardial infarction (MI).

48- 51. (Canceled)

52. (Currently amended) A method according to ~~any one of claims 47-51~~ claim 47, further comprising prophylactically administering an MI therapeutic agent to a human subject identified as having increased risk of developing MI, wherein the MI therapeutic agent inhibits leukotriene synthesis by inhibiting the activity of at least one protein selected from 5-Lipoxygenase activating protein (FLAP) and 5-lipoxygenase (5-LO).

53. (Original) A method of decreasing risk of a subsequent myocardial infarction in an individual who has had at least one myocardial infarction, comprising

administering a therapeutically effective amount of an MI therapeutic agent to the individual, wherein the MI therapeutic agent inhibits leukotriene synthesis by inhibiting the activity of at least one protein selected from 5-Lipoxygenase activating protein (FLAP) and 5-lipoxygenase (5-LO) and

monitoring myeloperoxidase (MPO) in the individual before and during the administration of the therapeutic agent, wherein the MI therapeutic agent is administered in an amount effective to reduce the MPO level in a subject.

54. (Original) A method of screening a human subject for susceptibility for MI comprising

analyzing nucleic acid of a human subject for the presence and absence of the FLAP haplotype comprised of markers:

SG13S377 (SEQ ID NO: 1, position 169965), allele A;

SG13S114 (SEQ ID NO: 1, position 178096), allele A;

SG13S41 (SEQ ID NO: 1, position 202045), allele A; and

SG13S35 (SEQ ID NO: 1, position 206117), allele G,

identifying the subject as having a susceptibility to MI, wherein the presence of the FLAP haplotype correlates with an increased risk of myocardial infarction.

55. (Original) A composition comprising a leukotriene synthesis inhibitor and a statin.

56-58. (Canceled)

59. (Currently amended) The composition according to ~~any one of claims 55-57~~ claim 55, wherein the leukotriene synthesis inhibitor is a FLAP inhibitor.

60-61. (Canceled)

62. (Original) The composition according to claim 59, wherein the FLAP inhibitor comprises BAY-X-1005 or a physiologically acceptable salt, formulation, or pro-drug thereof.

63. (Canceled)

64. (Currently amended) The composition according to ~~any one of claims 55-63~~ claim 55, wherein the statin is selected from the group consisting of rovuvastatin, fluvastatin, atorvastatin, lovastatin, simvastatin, pravastatin or pitavastatin.

65. (Currently amended) The composition according to ~~any one of claims 55-64~~ claim 55, wherein the leukotriene synthesis inhibitor is included in the composition in an amount effective to reduce serum C-reactive protein (CRP) in a human subject.

66. (Currently amended) The composition according to any ~~one of claims 55-65~~ claim 55 wherein the statin is included in the composition in an amount effective to reduce serum low density lipoprotein cholesterol (LDL) and reduce serum CRP in a human subject.

67. (Currently amended) The composition according to ~~any one of claims 55-65~~ claim 55, wherein the leukotriene inhibitor and the statin are included in the composition in amounts effective to synergistically reduce serum C-reactive protein in a human subject.

68-73. (Canceled)

74. (Currently amended) A method of reducing C reactive protein (CRP) in a human subject, comprising:

administering to a human in need of treatment to reduce CRP a composition according to ~~any one of claims 55-73~~ claim 55 in an amount effective to reduce serum C reactive protein in the human subject.

75. (Original) The method of claim 74, comprising:

selecting for the administering step a human subject at risk for a disease or condition selected from the group consisting of myocardial infarction, acute coronary syndrome, stroke, or peripheral arterial occlusive disease.

76. (Currently amended) The method of claim 74 ~~or 75~~, wherein the composition is administered in an amount effective to reduce serum LDL and serum leukotrienes in the human subject.

77. (Original) A method of reducing C reactive protein (CRP) in a human subject, comprising:

selecting a human subject that receives statin therapy to reduce serum LDL, wherein the statin therapy optionally reduces serum CRP in the human subject; and

administering to the human subject a leukotriene synthesis antagonist, in an amount effective to further reduce CRP in the human subject.

78. (Currently amended A method of reducing C reactive protein (CRP) in a human subject, comprising:

identifying a human subject in need of treatment to reduce serum CRP;

administering to the human subject a composition comprising a statin;

administering to the human subject a composition comprising a leukotriene synthesis inhibitor,

wherein the statin and the ~~leukotrience~~ leukotriene synthesis inhibitor are administered in amounts effective to reduce serum CRP in the human subject.

79-81. (Canceled)

82. (Currently amended) A method according to ~~any one of claims 78-81~~ claim 78, further comprising:

measuring serum C-reactive protein in the human subject to monitor therapeutic efficacy of the administering.

83. (Currently amended) A method according to ~~any one of claims 78-82~~ claim 78, further comprising modifying the amount or frequency of the administration following the measuring in order to achieve a target measurement of CRP in the human subject.

84. (Canceled)

85. (Previously presented) The method of claim 27, wherein, wherein the FLAP polymorphism further comprises marker SG13S25 (SEQ ID NO: 1, position 165553), allele G.

86. (Previously presented) The method of claim 85, wherein the FLAP polymorphism further comprises SG13S32 (SEQ ID NO: 1, position 176579), allele A.

87. (Previously presented) The method claim 86, wherein the FLAP polymorphism further comprises marker SG13S106 (SEQ ID NO: 1, position 198547), allele G or A.

88. (Previously presented) The method claim 30, wherein the haplotype further comprises marker SG13S25 (SEQ ID NO: 1, position 165553), allele G.

89. (Previously presented) The method claim 88, wherein the haplotype further comprises SG13S32 (SEQ ID NO: 1, position 176579), allele A.

90. (Previously presented) The method of claim 89, wherein the haplotype further comprises marker SG13S106 (SEQ ID NO: 1, position 198547), allele G or A.

91. (New) A method of assessing susceptibility to myocardial infarction (MI) or stroke in a human individual, the method comprising:

screening nucleic acid of the individual to determine whether the nucleic acid comprises a 5-lipoxygenase activating protein (FLAP) haplotype that comprises polymorphisms SG13S25, allele G; and SG13S114, allele T;

wherein the presence of the haplotype in the nucleic acid of the individual identifies the individual as having elevated susceptibility to MI, and wherein the absence of the haplotype in the nucleic acid of the individual identifies the individual as not having the elevated susceptibility to MI or stroke.

92. (New) The method of claim 91, comprising obtaining a biological sample from the individual that comprises the nucleic acid, and screening the nucleic acid from the biological sample.

93. (New) The method of claim 91, wherein the screening step comprises subjecting said nucleic acid to at least one procedure selected from the group consisting of: (a) enzymatic amplification of nucleic acid from the individual; (b) electrophoretic analysis; (c) restriction fragment length polymorphism analysis; and (d) nucleotide sequence analysis.

94. (New) A method of prophylaxis therapy for myocardial infarction (MI) comprising:

selecting a human subject having a FLAP (SEQ ID NO: 1) haplotype comprising: SG13S25 (SEQ ID NO: 1, position 165553), allele G; and SG13S114 (SEQ ID NO: 1, position 178096, allele T; and

administering to the subject a composition comprising a therapeutically effective amount of an MI therapeutic agent that inhibits leukotriene synthesis *in vivo*, wherein the MI therapeutic agent inhibits leukotriene synthesis.

95. (New) The method of claim 95, further comprising monitoring myeloperoxidase (MPO) level in the human subject before and during the prophylaxis treatment, wherein the MI therapeutic agent is administered in an amount effective to reduce the MPO level in a subject.